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Walnut Anthracnose

By Frederick H. Berry¹

Eastern black walnut (*Juglans nigra* L.) is one of our most valuable hardwood trees. It grows throughout most of the eastern half of the United States, generally as scattered trees in fields, along fence rows, or in hardwood stands. The wood is widely used for furniture, gunstocks, and cabinetmaking, and

buyers tour the countryside to bargain for individual trees. The distinctively flavored nuts are food for both humans and wildlife.

Walnut anthracnose, or leaf blotch as it is sometimes called, is a native disease that is particularly destructive on this walnut species throughout its range. It is caused by a fungus, *Gnomonia leptostyla* (Fr.) Ces. and deN., the imperfect stage of which is *Marssonina juglandis* (Lib.) Magn. The disease may quickly become epidemic dur-

¹ Forest pathologist, U.S. Department of Agriculture, Forest Service, Central States Forest Experiment Station, Forest Insect and Disease Laboratory, Delaware, Ohio.



Figure 1.—A black walnut tree almost completely defoliated by anthracnose.

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ing wet weather in the growing season and cause many walnut trees to lose almost all their leaves by late July or early August (fig. 1). This premature defoliation slows the trees' growth, weakens the trees greatly, and sometimes kills them. The wood of anthracnose-killed trees is not damaged, and prompt salvage will bring full market value.

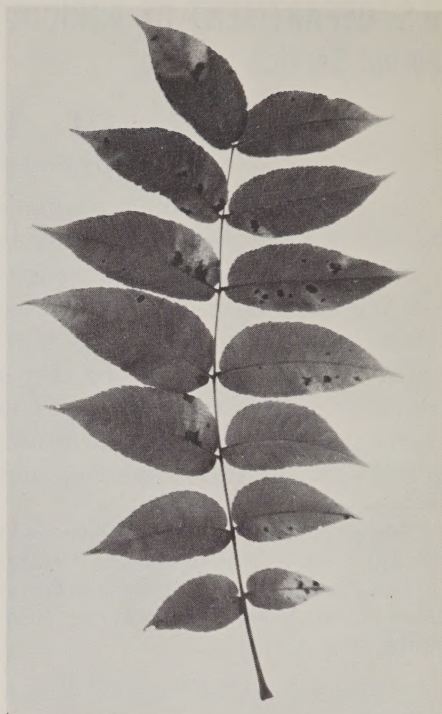
Hosts and Distribution

Individual eastern black walnut trees vary in their susceptibility to walnut anthracnose. But even the less susceptible ones become severely infected and defoliated when weather favors development of the fungus. Butternut (*Juglans cinerea* L.), Persian (English) walnut (*J. regia* L.), and first-generation hybrids of Persian and black walnut are also attacked. Two species of walnut native to California, the Hinds walnut (*J. hindsii* Jeps.) and the California walnut (*J. californica* S. Wats.), are both susceptible. Foliage of Persian and hybrid walnut trees is often severely infected with anthracnose during wet seasons, but defoliation is less than on eastern black walnut.

Walnut anthracnose is worldwide in distribution. Besides infecting *Juglans* species in the United States, the disease has been reported from most of the countries of Europe and from Argentina, Canada, and South Africa.

Symptoms

Leaves, nuts, and occasionally shoots of the current season's growth are all likely to be attacked. Tiny dark-brown or black spots, circular to irregularly circular in



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Figure 2.—Anthracnose spots on a black walnut leaf.

shape, appear on infected leaves (fig. 2). Gradually these spots become more numerous, enlarge, and often merge to form still larger dead areas. Yellowish to golden leaf tissue usually borders these spots. Infected leaves and leaflets generally fall prematurely, but some infected leaflets remain attached to the tree for most of the season.

Premature defoliation caused by the anthracnose fungus affects the quality and growth of nut meats. Nuts from diseased trees commonly have meats that are dark, unattractive, and shriveled. Sunken, necrotic spots, smaller than those on the leaves, appear on husks of infected nuts (fig. 3). Nuts that be-



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Figure 3.—Anthracnose spots on black walnut fruits.

come diseased when immature do not develop normally and many drop prematurely.

Lesions also appear on current shoots and form dead, sunken areas that are oval to irregularly circular in shape, light grayish brown in color, and dark reddish brown at their margins.

Life History of the Fungus

The fungus belongs to a group of fungi referred to as Ascomycetes because they bear the spores of the perfect or sexual stage in a club-shaped organ called an ascus. It usually overwinters in fallen walnut leaves infected during the preceding summer. Rarely, it overwinters in lesions on twigs of infected trees and in infected nuts on the ground. Primary infection occurs in the spring from ascospores. Discharged from the overwintered walnut leaves during rainy periods, the ascospores are carried upward by wind. If they lodge on a susceptible leaf under favorable conditions, the

ascospores germinate and leaf spots appear in about 14 to 16 days.

Secondary spores, the conidia, are produced on the diseased leaflets in minute black fruiting bodies or acervuli. Acervuli are abundant on the underside of leaflets, and occasionally a few are found on the upperside. Conidia are colorless, usually crescent shaped, and divided by a cross-wall into two approximately equal cells. They are borne in large numbers and are spread from leaf to leaf by wind and splashing rain. The rapid increase and spread of walnut anthracnose in the summer and fall is usually by means of repeated generations of conidia. Leaves are most likely to be infected and to fall off during wet weather.

Control

No practical control of anthracnose is known for walnut trees growing under forest conditions, although these trees are often severely infected.

For shade, ornamental, and nut-producing trees, one means of control is to destroy old leaves on the ground by raking and burning them. But this sometimes fails because fruiting bodies left behind on missed leaves or parts of leaves may release enough ascospores to cause primary infection.

Walnut anthracnose can be controlled where spraying is practicable. A protective fungicide such as zineb or maneb will provide effective control even during extremely wet seasons. These materials are applied at the rate of 2 pounds in 100 gallons of water or 1½ tablespoons per gallon. The first appli-

cation should be made when the leaves are $\frac{1}{2}$ to $\frac{3}{4}$ mature size to protect the trees from primary infection. Then two additional sprayings (three during wet seasons) need to be made at about 2-week intervals.

Other fungicides that have satisfactorily controlled anthracnose are bordeaux mixture, phenylmercury triethanol ammonium lactate (Puratized Agricultural Spray), and dodine.

Caution: Fungicides are poisonous and should be used with caution and according to recommendations of the manufacturer. They should be properly labeled and stored in a safe place away from food and children.

Reference

ETIOLOGY AND CONTROL OF WALNUT ANTHRACNOSE. Frederick H. Berry. Univ. Maryland Agr. Expt. Sta. Bul. A-113, 22 pp. 1960.

